

Connecting via Winsock to STN at pto-stn on port 23

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NEWS	10	NOV 22	Higher System Limits Increase the Power of STN Substance-Based Searching
NEWS	11	NOV 24	Search an additional 46,850 records with MEDLINE backfile extension to 1946
NEWS	12	DEC 14	New PNK Field Allows More Precise Crossover among STN Patent Databases
NEWS	13	DEC 18	ReaxysFile available on STN
NEWS	14	DEC 21	CAS Learning Solutions -- a new online training experience
NEWS	15	DEC 22	Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAPLUS
NEWS	16	JAN 24	The new and enhanced DPCI file on STN has been released
NEWS	17	JAN 26	Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents
NEWS	18	JAN 26	Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE
NEWS	19	JAN 28	CABA will be updated weekly
NEWS	20	FEB 23	PCTFULL file on STN completely reloaded
NEWS	21	FEB 23	STN AnaVist Test Projects Now Available for Qualified Customers
NEWS	22	FEB 25	LPCI will be replaced by LDPCI
NEWS	23	MAR 07	Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN
NEWS	24	APR 26	Expanded Swedish Patent Application Coverage in CA/CAPLUS Provides More Current and Complete Information
NEWS	25	APR 28	The DWPI (files WPINDEX, WPIDS and WPIX) on STN have been

enhanced with thesauri for the European Patent Classifications
 NEWS 26 MAY 02 MEDLINE Improvements Provide Fast and Simple Access to DOI and
 Chemical Name Information
 NEWS 27 MAY 12 European Patent Classification thesauri added to the INPADOC
 files, PCTFULL, GBFULL and FRFULL
 NEWS 28 MAY 20 PATDPA database updates to end in June 2011
 NEWS 29 MAY 23 STN biosequence searches with enhanced performance
 NEWS 30 MAY 23 Free Trial of the Numeric Property Search Feature
 in PCTFULL on STN

NEWS EXPRESS 17 DECEMBER 2010 CURRENT WINDOWS VERSION IS V8.4.2 .1,
 AND CURRENT DISCOVER FILE IS DATED 24 JANUARY 2011.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 10:06:00 ON 25 MAY 2011

```
=> file medline
COST IN U.S. DOLLARS          SINCE FILE      TOTAL
                              ENTRY      SESSION
FULL ESTIMATED COST          0.23      0.23
```

FILE 'MEDLINE' ENTERED AT 10:06:07 ON 25 MAY 2011

FILE LAST UPDATED: 24 May 2011 (20110524/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject
 Headings (MeSH) vocabulary and tree numbers from the U.S. National Library
 of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.html.

The 2011 Medline reload was completed on January 22, 2011.
 See HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate
 substance identification.

See HELP RANGE before carrying out any RANGE search.

```
=> s sequence requirements for micrna processing
    980083 SEQUENCE
    289417 SEQUENCES
    1065959 SEQUENCE
        (SEQUENCE OR SEQUENCES)
    113931 REQUIREMENTS
    8853374 FOR
    71 FORS
```

```

8853397 FOR
    (FOR OR FORS)
    8 MICRNA
    4 MICRNAS
    9 MICRNA
    (MICRNA OR MICRNAS)
306533 PROCESSING
    99 PROCESSINGS
306583 PROCESSING
    (PROCESSING OR PROCESSINGS)
L1      0 SEQUENCE REQUIREMENTS FOR MICRNA PROCESSING
        (SEQUENCE(W)REQUIREMENTS(W)FOR(W)MICRNA(W)PROCESSING)

=> s sequence requirements for microna processing
    980083 SEQUENCE
    289417 SEQUENCES
    1065959 SEQUENCE
        (SEQUENCE OR SEQUENCES)
    113931 REQUIREMENTS
    8853374 FOR
        71 FORS
    8853397 FOR
        (FOR OR FORS)
        17 MICRONA
    306533 PROCESSING
        99 PROCESSINGS
    306583 PROCESSING
        (PROCESSING OR PROCESSINGS)
L2      0 SEQUENCE REQUIREMENTS FOR MICRONA PROCESSING
        (SEQUENCE(W)REQUIREMENTS(W)FOR(W)MICRONA(W)PROCESSING)

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    289417 SEQUENCES
    1065959 SEQUENCE
        (SEQUENCE OR SEQUENCES)
    113931 REQUIREMENTS
    8853374 FOR
        71 FORS
    8853397 FOR
        (FOR OR FORS)
        62353 MICRO
        3518 MICROS
        65798 MICRO
        (MICRO OR MICROS)
    252867 NA
        3532 NAS
    255903 NA
        (NA OR NAS)
    306533 PROCESSING
        99 PROCESSINGS
    306583 PROCESSING
        (PROCESSING OR PROCESSINGS)
L3      0 SEQUENCE REQUIREMENTS FOR MICRO NA PROCESSING
        (SEQUENCE(W)REQUIREMENTS(W)FOR(W)MICRO(W)NA(W)PROCESSING)

=> s sequence requirements for micro rna processing
    980083 SEQUENCE
    289417 SEQUENCES
    1065959 SEQUENCE
        (SEQUENCE OR SEQUENCES)

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113931 REQUIREMENTS

8853374 FOR

71 FORS

8853397 FOR

(FOR OR FORS)

62353 MICRO

3518 MICROS

65798 MICRO

(MICRO OR MICROS)

628278 RNA

31663 RNAS

632468 RNA

(RNA OR RNAS)

306533 PROCESSING

99 PROCESSINGS

306583 PROCESSING

(PROCESSING OR PROCESSINGS)

L4 1 SEQUENCE REQUIREMENTS FOR MICRO RNA PROCESSING

(SEQUENCE (W) REQUIREMENTS (W) FOR (W) MICRO (W) RNA (W) PROCESSING)

=> d bib

L4 ANSWER 1 OF 1 MEDLINE on STN

AN 2003140447 MEDLINE <<LOGINID::20110525>>

DN PubMed ID: 12554881

TI Sequence requirements for micro RNA processing and function in human cells.

AU Zeng Yan; Cullen Bryan R

CS Howard Hughes Medical Institute, Department of Molecular Genetics and Microbiology, Duke University Medical Center, Durham, North Carolina 27710, USA.

SO RNA (New York, N.Y.), (2003 Jan) Vol. 9, No. 1, pp. 112-23.

Journal code: 9509184. ISSN: 1355-8382. L-ISSN: 1355-8382.

Report No.: NLM-PMC1370375.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200304

ED Entered STN: 27 Mar 2003

Last Updated on STN: 17 Apr 2003

Entered Medline: 16 Apr 2003

OSC.G 63 There are 63 MEDLINE records that cite this record

REM.CNT 29 There are 29 cited references available in MEDLINE for this document.

=> d his full

(FILE 'HOME' ENTERED AT 10:06:00 ON 25 MAY 2011)

FILE 'MEDLINE' ENTERED AT 10:06:07 ON 25 MAY 2011

L1 0 SEA PLU=ON SEQUENCE REQUIREMENTS FOR MICRNA PROCESSING

L2 0 SEA PLU=ON SEQUENCE REQUIREMENTS FOR MICRONA PROCESSING

L3 0 SEA PLU=ON SEQUENCE REQUIREMENTS FOR MICRO NA PROCESSING

L4 1 SEA PLU=ON SEQUENCE REQUIREMENTS FOR MICRO RNA PROCESSING

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FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 24 May 2011 (20110524/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at:

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The 2011 Medline reload was completed on January 22, 2011.
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This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> log y

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

1.04

1.27

STN INTERNATIONAL LOGOFF AT 10:07:26 ON 25 MAY 2011

Connecting via Winsock to STN at pto-stn on port 23

Welcome to STN International! Enter x:X

LOGINID:sssptal632ras

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

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FILE 'HOME' ENTERED AT 06:28:39 ON 26 MAY 2011

=> file medline

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.23	0.23

FILE 'MEDLINE' ENTERED AT 06:28:50 ON 26 MAY 2011

FILE LAST UPDATED: 25 May 2011 (20110525/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.html.

The 2011 Medline reload was completed on January 22, 2011.
See HELP RLOAD for details.

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See HELP RANGE before carrying out any RANGE search.

=> s (mirna or microrna or micro rna) response element
MISSING OPERATOR RNA) RESPONSE

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

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=> s (mirna or microrna or micro rna) (1a) response element
      5493 MIRNA
      5426 MIRNAS
      6704 MIRNA
            (MIRNA OR MIRNAS)
      6307 MICRORNA
     10014 MICRORNAS
     10846 MICRORNA
            (MICRORNA OR MICRORNAS)
     62372 MICRO
      3518 MICROS
     65817 MICRO
            (MICRO OR MICROS)
     628399 RNA
      31668 RNAS
     632588 RNA
            (RNA OR RNAS)
       505 MICRO RNA
            (MICRO(W)RNA)
    1469438 RESPONSE
     531522 RESPONSES
    1763118 RESPONSE
            (RESPONSE OR RESPONSES)
     114998 ELEMENT
     204235 ELEMENTS
     284466 ELEMENT
            (ELEMENT OR ELEMENTS)
     24035 RESPONSE ELEMENT
            (RESPONSE(W)ELEMENT)
L1          8 (MIRNA OR MICRORNA OR MICRO RNA) (1A) RESPONSE ELEMENT
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=> d bib ab 1-8

```
L1  ANSWER 1 OF 8      MEDLINE on STN
AN  2011350953      IN-PROCESS <<LOGINID::20110526>>
DN  PubMed ID: 21310851
TI  MicroRNA hsa-miR-613 targets the human LXR $\alpha$  gene and mediates a
    feedback loop of LXR $\alpha$  autoregulation.
AU  Ou Zhimin; Wada Taira; Gramignoli Roberto; Li Song; Strom Stephen C; Huang
```

Min; Xie Wen

CS Center for Pharmacogenetics, Department of Pharmaceutical Sciences,
University of Pittsburgh, Pittsburgh, Pennsylvania 15261, USA.

NC DK076962 (United States NIDDK NIH HHS)
ES014626 (United States NIEHS NIH HHS)

SO Molecular endocrinology (Baltimore, Md.), (2011 Apr) Vol. 25, No. 4, pp.
584-96. Electronic Publication: 2011-02-10.
Journal code: 8801431. E-ISSN: 1944-9917. L-ISSN: 0888-8809.
Report No.: NLM-PMC3063084 [Available on 04/01/12].

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals

ED Entered STN: 31 Mar 2011
Last Updated on STN: 13 May 2011

AB The nuclear receptor liver X receptor (LXR) is a ligand-dependent
transcription factor that plays an important role in the metabolism and
homeostasis of cholesterol, lipids, bile acids, and steroid hormones.
MicroRNAs (miRNAs) are recently recognized important negative regulators
of gene expression. In this report, we showed that miRNA hsa-miR-613
played an important role in the autoregulation of the human LXR α
gene. hsa-miR-613 targeted the endogenous LXR α through its specific
miRNA response element (613MRE) within the LXR α
3'-untranslated region. Interestingly and paradoxically, the expression
of hsa-miR-613 itself was induced upon the activation of LXR. However,
hsa-miR-613 did not appear to be a direct LXR target gene. Instead, the
positive regulation of hsa-miR-613 by LXR was mediated by the sterol
regulatory element binding protein (SREBP)-1c, a known LXR target gene.
Promoter analysis revealed an SREBP response element in the hsa-miR-613
gene promoter. Treatment with insulin also induced the expression of
hsa-miR-613 in an SREBP-1c-dependent manner, further supporting the role
of SREBP-1c in the positive regulation of this miRNA species. Finally,
the autoinduction of LXR α by a LXR agonist was enhanced when
hsa-miR-613 was inhibited or SREBP-1c was down-regulated. hsa-miR-613
appeared to specifically target the human LXR α . We propose that the
negative regulation mediated by hsa-miR-613 and SREBP-1c and the
previously reported positive regulation mediated by an LXR response
element in the LXR α gene promoter constitute a ying-yang mechanism
to ensure a tight regulation of this nuclear receptor of many metabolic
functions.

L1 ANSWER 2 OF 8 MEDLINE on STN

AN 2011201873 MEDLINE <<LOGINID::20110526>>

DN PubMed ID: 21219875

TI Breast cancer resistance protein BCRP/ABCG2 regulatory microRNAs
(hsa-miR-328, -519c and -520h) and their differential expression in
stem-like ABCG2+ cancer cells.

AU Li Xin; Pan Yu-Zhuo; Seigel Gail M; Hu Zi-Hua; Huang Min; Yu Ai-Ming

CS Department of Pharmaceutical Sciences, University at Buffalo, The State
University of New York, Buffalo, NY 14260-1200, USA.

NC R01 DA021172-04 (United States NIDA NIH HHS)
R01DA021172 (United States NIDA NIH HHS)
R21CA127061 (United States NCI NIH HHS)
U54CA143876 (United States NCI NIH HHS)

SO Biochemical pharmacology, (2011 Mar 15) Vol. 81, No. 6, pp. 783-92.
Electronic Publication: 2011-01-08.
Journal code: 0101032. E-ISSN: 1873-2968. L-ISSN: 0006-2952.
Report No.: NLM-NIHMS264683 [Available on 03/15/12]; NLM-PMC3042498
[Available on 03/15/12].

CY England: United Kingdom
 DT (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS Priority Journals
 EM 201104
 ED Entered STN: 22 Feb 2011
 Last Updated on STN: 19 Apr 2011
 Entered Medline: 18 Apr 2011
 AB Recent studies have shown that a number of microRNAs (miRNA or miR) may regulate human breast cancer resistance protein (BCRP/ABCG2), an important efflux transporter responsible for cellular drug disposition, whereas their effects on ABCG2 protein expression are not compared. In this study, we first identified a new proximal miRNA response element (MRE) for hsa-miR-519c within ABCG2 3'-untranslated region (3'UTR) through computational analyses. This miR-519c MRE site was confirmed using dual luciferase reporter assay and site-directed mutagenesis. Immunoblot analyses indicated that ABCG2 protein expression was significantly down-regulated in MCF-7/MX100 cells after transfection with hsa-miR-328- or -519c expression plasmids, and was markedly up-regulated in MCF-7 cells after transfection with miR-328 or -519c antagomir. However, ABCG2 protein expression was unchanged in MCF-7/MX100 cells after transfection with hsa-miR-520h expression plasmids, which was associated with undetectable miR-520h expression. Furthermore, ABCG2 mRNA degradation was accelerated dramatically in cells transfected with miR-519c expression plasmid, suggesting the involvement of mRNA degradation mechanism. Intervention of miR-328 or -519c signaling led to significant change in intracellular mitoxantrone accumulation, as determined by flow cytometry analyses. In addition, we separated RB143 human retinoblastoma cells into stem-like (ABCG2+) and non-stem-like (ABCG2-) populations through immunomagnetic selection, and found that miR-328, -519c and -520h levels were 9-, 15- and 3-fold lower in the ABCG2+ cells, respectively. Our data suggest that miR-519c and -328 have greater impact on ABCG2 expression than miR-520h in MCF-7 human breast cancer cells, and the presence of proximal miR-519c MRE explains the action of miR-519c on shortened ABCG2 3'UTR.
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L1 ANSWER 3 OF 8 MEDLINE on STN
 AN 2011141520 IN-PROCESS <<LOGINID::20110526>>
 DN PubMed ID: 21264258
 TI Role of microRNA-26b in glioma development and its mediated regulation on EphA2.
 AU Wu Ning; Zhao Xiangzhong; Liu Ming; Liu Haizhou; Yao Weicheng; Zhang Yuyan; Cao Shousong; Lin Xiukun
 CS Institute of Oceanology, Chinese Academy of Sciences, Qingdao, China.
 SO PloS one, (2011) Vol. 6, No. 1, pp. e16264. Electronic Publication: 2011-01-14.
 Journal code: 101285081. E-ISSN: 1932-6203. L-ISSN: 1932-6203.
 Report No.: NLM-PMC3021542.

CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
 ED Entered STN: 26 Jan 2011
 Last Updated on STN: 17 Feb 2011
 AB BACKGROUND: MicroRNAs (miRNAs) are short, non-coding RNAs that regulate the expression of multiple target genes. Deregulation of miRNAs is common

in human tumorigenesis. Low level expression of miR-26b has been found in glioma cells. However, its underlying mechanism of action has not been determined.

METHODOLOGY/PRINCIPAL FINDINGS: Real-time PCR was employed to measure the expression level of miR-26b in glioma patients and cells. The level of miR-26b was inversely correlated with the grade of glioma. Ectopic expression of miR-26b inhibited the proliferation, migration and invasion of human glioma cells. A binding site for miR-26b was identified in the 3'UTR of EphA2. Over-expression of miR-26b in glioma cells repressed the endogenous level of EphA2 protein. Vasculogenic mimicry (VM) experiments were performed to further confirm the effects of miR-26b on the regulation of EphA2, and the results showed that miR-26b inhibited the VM processes which regulated by EphA2.

SIGNIFICANCE: This study demonstrated that miR-26b may act as a tumor suppressor in glioma and it directly regulates EphA2 expression. EphA2 is a direct target of miR-26b, and the down-regulation of EphA2 mediated by miR-26b is dependent on the binding of miR-26b to a specific response element of microRNA in the 3'UTR region of EphA2 mRNA.

L1 ANSWER 4 OF 8 MEDLINE on STN
AN 2009633266 MEDLINE <<LOGINID::20110526>>
DN PubMed ID: 19581388
TI MicroRNAs regulate CYP3A4 expression via direct and indirect targeting.
AU Pan Yu-Zhuo; Gao Wenqing; Yu Ai-Ming
CS Department of Pharmaceutical Sciences, School of Pharmacy and
Pharmaceutical Sciences, University at Buffalo, The State University of
New York, Buffalo, NY 14260-1200, USA.
NC R01-DA021172 (United States NIDA NIH HHS)
SO Drug metabolism and disposition: the biological fate of chemicals, (2009
Oct) Vol. 37, No. 10, pp. 2112-7. Electronic Publication: 2009-07-06.
Journal code: 9421550. E-ISSN: 1521-009X. L-ISSN: 0090-9556.
Report No.: NLM-PMC2769037.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 201001
ED Entered STN: 22 Sep 2009
Last Updated on STN: 19 Jan 2010
Entered Medline: 18 Jan 2010
AB CYP3A4 metabolizes many drugs on the market. Although transcriptional
regulation of CYP3A4 is known to be tightly controlled by some nuclear
receptors (NR) including vitamin D receptor (VDR/NR1I1),
posttranscriptional regulation of CYP3A4 remains elusive. In this study,
we show that noncoding microRNAs (miRNAs) may control posttranscriptional
and transcriptional regulation of CYP3A4 by directly targeting the
3'-untranslated region (3'UTR) of CYP3A4 and indirectly targeting the
3'UTR of VDR, respectively. Luciferase reporter assays showed that CYP3A4
3'UTR-luciferase activity was significantly decreased in human embryonic
kidney 293 cells transfected with plasmid that expressed microRNA-27b
(miR-27b) or mouse microRNA-298 (mmu-miR-298), whereas the activity was
unchanged in cells transfected with plasmid that expressed microRNA-122a
or microRNA-328. Disruption of the corresponding miRNA response
element (MRE) within CYP3A4 3'UTR led to a 2- to 3-fold increase in
luciferase activity. Immunoblot analyses indicated that CYP3A4 protein
was down-regulated over 30% by miR-27b and mmu-miR-298 in LS-180 and PANC1
cells. The decrease in CYP3A4 protein expression was associated with

significantly decreased CYP3A4 mRNA levels, as determined by quantitative real-time PCR (qPCR) analyses. Likewise, interactions of miR-27b or mmu-miR-298 with VDR 3'UTR were supported by luciferase reporter assays. The mmu-miR-298 MRE site is well conserved within the 3'UTR of mouse, rat, and human VDR. Down-regulation of VDR by the two miRNAs was supported by immunoblot and qPCR analyses. Furthermore, overexpression of miR-27b or mmu-miR-298 in PANC1 cells led to a lower sensitivity to cyclophosphamide. Together, these findings suggest that CYP3A4 gene expression may be regulated by miRNAs at both the transcriptional and posttranscriptional level.

L1 ANSWER 5 OF 8 MEDLINE on STN
AN 2009614469 MEDLINE <<LOGINID::20110526>>
DN PubMed ID: 19635812
TI MicroRNA-125b promotes neuronal differentiation in human cells by repressing multiple targets.
AU Le Minh T N; Xie Huangming; Zhou Beiyan; Chia Poh Hui; Rizk Pamela; Um Moonkyoung; Udolph Gerald; Yang Henry; Lim Bing; Lodish Harvey F
CS Whitehead Institute for Biomedical Research, 9 Cambridge Center, Suite 601, Cambridge, MA 02142, USA.
NC AI54973 (United States NIAID NIH HHS)
DK047636 (United States NIDDK NIH HHS)
R01 DK068348 (United States NIDDK NIH HHS)
SO Molecular and cellular biology, (2009 Oct) Vol. 29, No. 19, pp. 5290-305. Electronic Publication: 2009-07-27.
Journal code: 8109087. E-ISSN: 1098-5549. L-ISSN: 0270-7306.
Report No.: NLM-PMC2747988.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(VALIDATION STUDIES)
LA English
FS Priority Journals
EM 200910
ED Entered STN: 12 Sep 2009
Last Updated on STN: 3 Oct 2009
Entered Medline: 2 Oct 2009
OSC.G 1 There are 1 MEDLINE records that cite this record
REM.CNT 41 There are 41 cited references available in MEDLINE for this document.
AB MicroRNAs (miRNAs) are a class of small noncoding RNAs that regulate gene expression at the posttranscriptional level. Research on miRNAs has highlighted their importance in neural development, but the specific functions of neurally enriched miRNAs remain poorly understood. We report here the expression profile of miRNAs during neuronal differentiation in the human neuroblastoma cell line SH-SY5Y. Six miRNAs were significantly upregulated during differentiation induced by all-trans-retinoic acid and brain-derived neurotrophic factor. We demonstrated that the ectopic expression of either miR-124a or miR-125b increases the percentage of differentiated SH-SY5Y cells with neurite outgrowth. Subsequently, we focused our functional analysis on miR-125b and demonstrated the important role of this miRNA in both the spontaneous and induced differentiations of SH-SY5Y cells. miR-125b is also upregulated during the differentiation of human neural progenitor ReNcell VM cells, and miR-125b ectopic expression significantly promotes the neurite outgrowth of these cells. To identify the targets of miR-125b regulation, we profiled the global changes in gene expression following miR-125b ectopic expression in SH-SY5Y cells. miR-125b represses 164 genes that contain the seed match sequence of the miRNA and/or that are predicted to be direct targets of miR-125b by conventional methods. Pathway analysis suggests that a subset of

miR-125b-repressed targets antagonizes neuronal genes in several neurogenic pathways, thereby mediating the positive effect of miR-125b on neuronal differentiation. We have further validated the binding of miR-125b to the miRNA response elements of 10 selected mRNA targets. Together, we report here for the first time the important role of miR-125b in human neuronal differentiation.

L1 ANSWER 6 OF 8 MEDLINE on STN
 AN 2009405411 MEDLINE <<LOGINID::20110526>>
 DN PubMed ID: 19483680
 TI MicroRNA-mediated species-specific attenuation of influenza A virus.
 AU Perez Jasmine T; Pham Alissa M; Lorini Maria H; Chua Mark A; Steel John; tenOever Benjamin R
 CS Microbiology Graduate School Training Program, Mount Sinai School of Medicine, New York, New York, USA.
 NC T32AI007647-09 (United States NIAID NIH HHS)
 SO Nature biotechnology, (2009 Jun) Vol. 27, No. 6, pp. 572-6. Electronic Publication: 2009-05-31.
 Journal code: 9604648. E-ISSN: 1546-1696. L-ISSN: 1087-0156.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 (RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
 LA English
 FS Priority Journals
 EM 200909
 ED Entered STN: 11 Jun 2009
 Last Updated on STN: 30 Sep 2009
 Entered Medline: 29 Sep 2009
 OSC.G 1 There are 1 MEDLINE records that cite this record
 AB Influenza A virus leads to yearly epidemics and sporadic pandemics. Present prophylactic strategies focus on egg-grown, live, attenuated influenza vaccines (LAIVs), in which attenuation is generated by conferring temperature sensitivity onto the virus. Here we describe an alternative approach to attenuating influenza A virus based on microRNA-mediated gene silencing. By incorporating nonavian microRNA response elements (MREs) into the open-reading frame of the viral nucleoprotein, we generate reassortant LAIVs for H1N1 and H5N1 that are attenuated in mice but not in eggs. MRE-based LAIVs show a greater than two-log reduction in mortality compared with control viruses lacking MREs and elicit a diverse antibody response. This approach might be combined with existing LAIVs to increase attenuation and improve vaccine safety.

L1 ANSWER 7 OF 8 MEDLINE on STN
 AN 2009251472 MEDLINE <<LOGINID::20110526>>
 DN PubMed ID: 19293287
 TI MicroRNA-125b is a novel negative regulator of p53.
 AU Le Minh T N; Teh Cathleen; Shyh-Chang Ng; Xie Huangming; Zhou Beiyan; Korzh Vladimir; Lodish Harvey F; Lim Bing
 CS Computation and Systems Biology, Singapore-Massachusetts Institute of Technology Alliance, Singapore.
 NC AI54973 (United States NIAID NIH HHS)
 DK47636 (United States NIDDK NIH HHS)
 R01 DK068348 (United States NIDDK NIH HHS)
 SO Genes & development, (2009 Apr 1) Vol. 23, No. 7, pp. 862-76. Electronic Publication: 2009-03-17.
 Journal code: 8711660. E-ISSN: 1549-5477. L-ISSN: 0890-9369.
 Report No.: NLM-PMC2666337.
 CY United States

DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 200904

ED Entered STN: 3 Apr 2009

Last Updated on STN: 16 Apr 2009

Entered Medline: 15 Apr 2009

OSC.G 8 There are 8 MEDLINE records that cite this record

REM.CNT 51 There are 51 cited references available in MEDLINE for this document.

AB The p53 transcription factor is a key tumor suppressor and a central regulator of the stress response. To ensure a robust and precise response to cellular signals, p53 gene expression must be tightly regulated from the transcriptional to the post-translational levels. Computational predictions suggest that several microRNAs are involved in the post-transcriptional regulation of p53. Here we demonstrate that miR-125b, a brain-enriched microRNA, is a bona fide negative regulator of p53 in both zebrafish and humans. miR-125b-mediated down-regulation of p53 is strictly dependent on the binding of miR-125b to a microRNA response element in the 3' untranslated region of p53 mRNA. Overexpression of miR-125b represses the endogenous level of p53 protein and suppresses apoptosis in human neuroblastoma cells and human lung fibroblast cells. In contrast, knockdown of miR-125b elevates the level of p53 protein and induces apoptosis in human lung fibroblasts and in the zebrafish brain. This phenotype can be rescued significantly by either an ablation of endogenous p53 function or ectopic expression of miR-125b in zebrafish. Interestingly, miR-125b is down-regulated when zebrafish embryos are treated with gamma-irradiation or camptothecin, corresponding to the rapid increase in p53 protein in response to DNA damage. Ectopic expression of miR-125b suppresses the increase of p53 and stress-induced apoptosis. Together, our study demonstrates that miR-125b is an important negative regulator of p53 and p53-induced apoptosis during development and during the stress response.

L1 ANSWER 8 OF 8 MEDLINE on STN

AN 2007527050 MEDLINE <<LOGINID::20110526>>

DN PubMed ID: 17689888

TI Comparative analysis of the SBP-box gene families in *P. patens* and seed plants.

AU Riese Maike; Hohmann Susanne; Saedler Heinz; Munster Thomas; Huijser Peter
CS Max Planck Institute for Plant Breeding Research, Cologne, Germany.

SO Gene, (2007 Oct 15) Vol. 401, No. 1-2, pp. 28-37. Electronic Publication: 2007-07-10.

Journal code: 7706761. ISSN: 0378-1119. L-ISSN: 0378-1119.

CY Netherlands

DT (COMPARATIVE STUDY)

Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200710

ED Entered STN: 11 Sep 2007

Last Updated on STN: 25 Oct 2007

Entered Medline: 24 Oct 2007

OSC.G 6 There are 6 MEDLINE records that cite this record

AB To come to a better understanding of the evolution and function of the SBP-box transcription factor family in plants, we identified, isolated and characterized 13 of its members from the moss *Physcomitrella patens*. For the majority of the moss SBP-box genes, clear orthologous relationships with family members of flowering plants could be established by

phylogenetic analysis based on the conserved DNA-binding SBP-domain, as well as additional synapomorphic molecular characters. The *P. patens* SBP-box genes cluster in four separable groups. One of these consists exclusively of moss genes; the three others are shared with family members of *Arabidopsis* and rice. Besides the family defining DNA-binding SBP-domain, other features can be found conserved between moss and other plant SBP-domain proteins. An AHA-like motif conserved from the unicellular alga *Chlamydomonas reinhardtii* to flowering plants, was found able to promote transcription in a heterologous yeast system. The conservation of a functional microRNA response element in the mRNA of three of the moss SBP-box genes supports the idea of an ancient origin of microRNA dependent regulation of SBP-box gene family members. As our current knowledge concerning the roles of SBP-box genes in plant development is scarce and the model system *P. patens* allows targeted mutation, the material we isolated and characterized will be helpful to generate the mutant phenotypes necessary to further elucidate these roles.

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FILE 'MEDLINE' ENTERED AT 06:28:50 ON 26 MAY 2011

L1 8 SEA PLU=ON (MIRNA OR MICRORNA OR MICRO RNA) (1A) RESPONSE
ELEMENT
D BIB AB 1-8

FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 25 May 2011 (20110525/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.

The 2011 Medline reload was completed on January 22, 2011.
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